

Congenital malaria in infants of asymptomatic women

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Congenital malaria accounts for 0.3% of all cases of malaria in endemic areas.¹ It usually occurs in infants of women who have had clinical attacks of malaria during pregnancy but also occurs, rarely, in infants of women who were asymptomatic throughout pregnancy.²⁻⁴ With the changing profile of immigrants to North America the incidence of malaria is increasing: 515 cases were reported in Canada in 1987 compared with 65 cases in 1975 and 254 in 1984.^{5,6} As a result, physicians must be more aware of the many manifestations of this infection, which can cause significant rates of illness and death. We present case reports of two infants with congenital malaria born to asymptomatic immigrant women, only the third such report in North America.^{2,4}

Case reports

Case 1

A 28-day-old boy was admitted to hospital in January 1991 with a 48-hour history of fever and irritability. The results of a physical examination were unremarkable except for hepatosplenomegaly. The initial hemoglobin level was 125 g/L, the leukocyte count was $4.0 \times 10^9/L$, with a normal differential count, and the platelet count was $45 \times 10^9/L$. The blood film revealed *Plasmodium vivax* parasites in red blood cells.

The mother was an East Indian woman who had moved to Canada in December of 1989, a year before her delivery. After the baby's condition was

diagnosed she revealed that she had been treated for malaria in India in 1988. She had subsequently been well except for 2 days of fever in early 1989 — apparently a condition not associated with malaria — for which she received medications. She had been totally asymptomatic during her pregnancy and had remained so after the delivery. The pregnancy and labour had been uneventful. The infant's birth weight had been 2812 g. The Apgar score had been only 2 at 1 minute but had improved to 6 at 5 minutes and 9 at 10 minutes. There was no obvious explanation for the initial low score. The infant had subsequently done well and was discharged home on day 5 of life. After the baby's malaria was diagnosed a review of the mother's blood film obtained a month earlier during routine admission for her labour and delivery revealed that less than 1% of her red cells contained *P. vivax* parasites.

Screening for glucose-6-phosphate dehydrogenase in mother and child gave normal results. The infant was treated for 48 hours with chloroquine, and the mother received chloroquine followed by primaquine. The infant's liver and spleen remained palpable. The platelet count increased to $280 \times 10^9/L$ and the leukocyte count to $18.8 \times 10^9/L$ with 81% lymphocytes. At the 2-month follow-up visit the infant was still well, and there was no further evidence of hepatosplenomegaly.

Case 2

A 22-day-old girl with a 24-hour history of fever and irritability was seen in the emergency depart-

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ment in May 1991. The initial hemoglobin level was 112 g/L, the leukocyte count was $10 \times 10^9/L$, with an increased proportion of lymphocytes, and the platelet count was $44 \times 10^9/L$. The blood film revealed *P. vivax* parasites in red blood cells.

The mother was a Vietnamese woman who had moved to Canada in September of 1989. She had been treated for malaria in Vietnam in early 1988 and had since been totally asymptomatic. The pregnancy and labour had been uneventful. The infant's birth weight had been 3300 g and the Apgar scores 9 at 1 minute and 9 at 5 minutes.

Screening for glucose-6-phosphate dehydrogenase gave normal results. Both infant and mother were treated with chloroquine, and the mother also received primaquine. The infant's platelet count increased to $398 \times 10^9/L$ with treatment, and she was well at her 1-month follow-up visit.

Comments

These case reports are instructive in that the

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diagnosis of malaria was missed in asymptomatic women from areas in which the condition is endemic; thus, there was a danger to their unborn children. Congenital malaria can develop with any of the species of *Plasmodium* but most commonly is due to *falciparum* and *vivax*. Maternal infections with *P. ovale* and *vivax* may be prolonged if the dormant hepatic phase is not eradicated adequately with primaquine. *P. falciparum* and *P. malariae* may persist as subclinical infections in the blood stream of untreated, partially immune people for long periods — up to 40 years. In most children with congenital malaria the infection develops at birth, because of the exchange of maternal and fetal blood.

These case reports demonstrate many of the typical features of congenital malaria. The child is usually healthy at birth, and symptoms appear at 3 to 12 weeks of age as the level of passively acquired maternal antibodies starts to wane. Fever is most commonly accompanied by irritability, hepatosplenomegaly, anorexia, progressive hemolytic anemia and thrombocytopenia.⁷ Treatment of congenital vivax malaria consists of oral administration of chloroquine (10 mg/kg of base followed by 5 mg/kg of base at 6, 24 and 48 hours). Primaquine is not needed for the newborn infant, because dormant hepatic foci do not develop. As the number of immigrants from Asia and Africa continues to increase, congenital malaria should be considered in the differential diagnosis of unexplained fever in infants born to women from these areas.

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